CHAPTER 15

Physiology of Sleep-Disordered Breathing

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CHAPTER OUTLINE

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OBJECTIVES

After reading this chapter, you will be able to:

- Explain how the brain regulates sleeping and waking cycles through neurocontrolled chemical mediators
- Explain why the upper airway anatomy plays such a major role in sleep-disordered breathing (SDB)
- Distinguish between the three stages of nonrapid eye movement sleep and rapid eye movement sleep
- Identify the six major categories of sleep disorders and the specific classification for SDB
- Differentiate between obstructive sleep apnea, central sleep apnea, mixed sleep apnea, childhood sleep apnea, and sudden infant death syndrome
- Explain the effects of SDB on the cardiovascular system
- Identify the mechanisms of central hemodynamic dysfunction secondary to SDB
- Classify SDB through the use of the polysomnogram
- Explain the physiological treatment of SDB using positive airway pressure, oral appliances, positional therapy, and airway enlargement treatment

KEY TERMS

alpha waves
apnea
apnea-hypopnea index (AHI)
bilevel positive airway pressure (BIPAP)
central sleep apnea (CSA)
circadian rhythm
congestive heart failure
continuous positive airway pressure (CPAP)
delta waves
durable medical equipment (DME)
dyssomnias
electroencephalogram (EEG)
electromyography (EMG)
extensive daytime somnolence
heart failure
hypersomnolence
hypopnea
hypothalamus
insomnia
macroglossia
Mallampati score
mandibular advancement device (MAD)
maxillomandibular advancement
micrognathia
mixed sleep apnea (MSA)
montage
nasal cycling
nonrapid eye movement (NREM)
obstructive sleep apnea (OSA)
polysomnogram (PSG)
The study of sleep has intrigued people through the ages. With more than one third of the human life span spent sleeping, the mystery of sleep and dreams is an experience common to everyone; yet, research has yielded few answers to the secrets of sleep. Although documentation of sleep disorders and dreaming can be found throughout recorded history, scientists have yet to discover the ultimate reason why humans need sleep, how it directly affects waking functionality, and the nature of physiological mechanisms associated with sleep. Certain anatomical and physiological disorders commonly associated with sleep-disordered breathing (SDB) are better understood; these include abnormal respiratory patterns and poor ventilation quality during sleep. Key to the understanding of SDB is a basic comprehension of the brain’s role in controlling sleep and the anatomical and physiological features of the upper airways.

**Neurocontrol of Sleep**

Sleep has been described as “a reversible behavioral state of perceptual unresponsiveness to the environment with documentable, distinguishable physiological patterns of neural and muscle activity.” The diagnostic test performed to evaluate the quality of sleep is called a sleep study, or polysomnogram (PSG). The PSG documents more than 20 individual physiological parameters during all phases of the sleep cycle. The term polysomnogram is derived from its prefix, root, and suffix definitions: “poly” means “many,” “somno” means “sleep,” and “gram” means “writing.” This quantitative diagnostic study records physiological data during each second of sleep from wakefulness through the three sleep stages of nonrapid eye movement (NREM) to the rapid eye movement (REM) stage. Through the PSG, the sleep process can be classified in an objective, quantifiable way. However, the precise mechanisms responsible for sleep onset and waking from sleep continue to be the focus of intensive investigation. Although the brain plays a definitive role in sleep regulation, the specific anatomical region of the brain responsible for the sleep control is a subject of controversy. It is now well established that the hypothalamus is the key regulator of sleep and wakefulness. Since sleep research is now focused on the hypothalamus, new pharmaceutical and therapeutic breakthroughs for neurological sleep disorders have been developed.

The brain is composed of five major regions: the medulla oblongata, which is the most posterior portion of the brain that fuses with the spinal cord; the pons, located superiorly to the medulla oblongata; the midbrain or mesencephalon, located just above the pons; the telencephalon or cerebrum, which consists of five paired lobes within two convoluted hemispheres; and the diencephalon, which sits superior to the brainstem nestled between the two cerebral hemispheres. The diencephalon includes the thalamus, the hypothalamus, the optic chiasm, and the pineal gland (Figure 15-1). Sleep researchers have identified more than 70 distinct sleep disorders but have yet to identify the precise neurochemical mechanisms that “turn on and turn off” sleep and wakefulness. As mentioned, the central focus of study in sleep regulation is currently the hypothalamus.

Biomedical sleep research has definitively determined that the primary biological “sleep clock” resides in the suprachiasmatic nucleus (SCN) of the hypothalamus.
nucleus (SCN) of the hypothalamus where it controls the timing of the sleep cycle, or the circadian rhythm. When SCN function is disrupted, the circadian rhythm becomes random and sporadic. However, even though the onset of sleep may vary throughout a 24-hour period, the total sleep time remains the same. Located just above the optic chiasm, the SCN is believed to control certain neurologically secreted chemicals that regulate the sleep cycle. Light plays a very important role in the normal regulation of the SCN. Light is an environmental cue or stimulator known as the zeitgeber, or “time-giver,” that regulates the sleep/wake cycle. The role that light plays as a circadian rhythm “cue” can be observed through the disrupted sleep habits of individuals living in geographical areas of prolonged winter darkness. A decrease in the amount of daylight exposure significantly alters the normal sleep/wake cycle during the winter seasons. The optic chiasm is thought to relay light/dark cues from the eye to the hypothalamus as a reference for the circadian rhythm. It is now understood that the SCN of the hypothalamus merely tracks day and night cycles, whereas the ventrolateral preoptic nucleus (VLPO) in the hypothalamus initiates sleep by inhibiting the brain’s arousal centers in the brainstem (Figure 15-2).

The role of the hypothalamus in controlling the regulation of sleep/wake mechanisms has been confirmed. Various neurotransmitters released by the hypothalamus are believed to produce sleep by inhibiting the brain’s arousal centers. The neurotransmitters that mediate arousal in the cortex include histamine, serotonin, norepinephrine, acetylcholine, and hypocretin (orexin) (Figure 15-3). Sleep research has shown that disruption of the VLPO and the lateral region of the hypothalamus results in arousal. The loss of the VLPO and the lateral hypothalamus structures have been associated with the symptoms of insomnia or narcolepsy respectively.

**CONCEPT QUESTION 15-1**

What part of the brain has been shown to be the primary controller for sleep and wakefulness?

**UPPER AIRWAY ANATOMY AND SLEEP**

Human neonates have the remarkable ability to breathe, suck, and swallow simultaneously owing to the anatomical configuration of the upper airway at birth. Additionally, neonates are obligatory nose breathers. During the first month of life, the neonatal upper airway grows and matures toward adult upper airway proportions; the infant’s ability to breathe, suck, and swallow simultaneously wanes. Throughout infancy, adolescence, and
adulthood, humans remain obligate nose breathers during sleep, which is often the source of significant problems for individuals with sleep disorders.

Oropharynx

The upper airways comprise three primary anatomical regions: the nose, pharynx, and larynx. As discussed in Chapter 1, normal pharyngeal muscle tone prevents the base of the tongue from falling backward in the oral cavity and obstructing the oropharyngeal airway. Sleep or unconscious states result in a loss of pharyngeal muscle tone and the relaxation of the soft tissue that constitutes the oropharynx and the tongue. Under the influence of gravity, the relaxed tongue falls into the oropharynx, partially or totally occluding the airway. Partial occlusions result in low-pitched snoring sounds, whereas complete occlusion results in apnea, characterized by ventilatory efforts without airflow. Several other soft tissue anatomical structures in the oropharynx may contribute to partial or total obstruction.

Obligate nose breathing during sleep causes problems when the nose is obstructed. Many individuals experience difficulty sleeping when nose breathing is limited because of nasal congestion that accompanies a cold or allergy. At the onset of sleep when obligate nose breathing begins, restricted nasal air passages usually cause sleepless or restless nights. Anatomical abnormalities such as a deviated septum create a permanent limitation of airflow through one or both of the nares. Poor quality of sleep is often a major complaint of individuals with nasal septum abnormalities. The assessment of a potential sleep disorder should always begin with inspection of each naris for patency and unrestricted airflow. If allergies are an issue, medications that diminish nasal swelling or secretions to reduce airflow restriction should be considered. If anatomical obstruction is the primary cause of airflow limitations, corrective surgery may be the best remedy for the sleep disorder. If the nose is occluded during sleep, secondary mouth breathing becomes necessary. Regardless of nose or mouth breathing, the oropharyngeal airway must remain patent to allow ventilation.

Within the oral cavity, five specific structures can obstruct airflow. Mallampati was the first to categorize the amount of “open space” in the oropharynx by the visualization of five structures: the tongue, the soft palate, the hard palate, the uvula, and the tonsils. Originally used to classify the difficulty level of oral endotracheal intubation, the Mallampati score is determined by direct visualization of the oropharynx through the open mouth. There are four categories of decreasing airway space (Figure 15-4). A class 1 score is considered normal, in which all five anatomical structures are visible. In class 2, all five structures can be identified, but only the upper portions of the tonsils and uvula are visible. Class 3 allows only the tongue, the soft and hard palate, and the base of the uvula to be seen. Class 4 allows visualization of only the hard palate and tongue. The higher the Mallampati classification number, the more anatomical crowding with less oropharyngeal room for airflow. Sleep research has shown a positive correlation between a high Mallampati score and the risk for obstructive sleep disorders when nasal obstruction is present.

Because the mandible anatomically supports and positions the tongue and is part of the oropharyngeal structure, the size of the mandible may limit airflow during sleep. A small, recessed lower jaw, or retrognathia, results in a more retrograde or posteriorly positioned tongue. A posterior position of the tongue makes it easier for the tongue to fall back and
block the oropharynx as the pharyngeal muscles relax. An enlarged tongue, or macroglossia, as seen in individuals with Down syndrome, hypothyroidism, and acromegaly, can also crowd the oropharynx decreasing the size of the airway lumen. A shortened or widened neck may also crowd the anatomical structures, narrowing or obstructing the oropharyngeal lumen. A neck circumference greater than 17 inches in men or greater than 16 inches in women is highly correlated with the incidence of obstructive sleep apnea (OSA). 

**CONCEPT QUESTION 15-2**

*During sleep, humans become obligate nose breathers. How does the oropharyngeal anatomy affect breathing during sleep?*

**CONCEPT QUESTION 15-3**

*How is the Mallampati score used to assess the possible presence of SDB?*

**STAGES OF SLEEP**

Sleep is an active process in which specific regions of the brain show continuous electrical activity that can be physiologically monitored. An understanding of the normal stages of sleep is necessary for the clinician to evaluate sleep disorders. In adults and children, the two major states of sleep are non-rapid eye movement (NREM) and rapid eye movement (REM). Sleep stages are categorized according to the absence or presence of eye movement.

The normal pattern of sleep stages involves the cycling back and forth between NREM and REM stages every 60 to 90 minutes for four to five cycles during an 8-hour sleep period. Sleep normally begins with the NREM stages and progresses to REM sleep. During the awake phase, the electroencephalogram (EEG), or electrical waveform of brain activity, shows a pattern of small, fine waves oscillating at a high frequency, known as alpha waves (Figure 15-5). At the onset of sleep, alpha waveforms slow and change to waveforms characteristic of NREM sleep, suggesting a resting or restorative state of the brain.

**Nonrapid Eye Movement**

NREM sleep is composed of three stages with each progressive stage transitioning to a deeper state of sleep. Stage 1 NREM represents the onset of sleep for adults and children. During stage 1 NREM, the eyes roll gently and slowly while low-amplitude EEG brainwaves are noted during the sleep study. This change in EEG brain wave pattern is associated with the transition from awake to sleep. Only 5% to 10% of the entire sleep period is spent in stage 1 NREM, and within 2 to 10 minutes, sleep
usually progresses to stage 2 NREM. EEG tracings show sharp spikes called “K-complexes” and “sleep spindles,” which serve as markers for the transition from stage 1 NREM to stage 2 NREM. In adults, approximately 40% to 50% of the total sleep period is spent in stage 2 NREM.  

Stage 3 NREM is the deepest stage of sleep and represents approximately 25% of the sleep period. The EEG displays delta waves, or slow-wave sleep, characterized by high-amplitude waves. “Delta sleep” is thought to represent restorative sleep, during which the brain is in its deepest state of sleep. Stage 3 NREM is characterized by a very low level of patient responsiveness; it is difficult to awaken a person from this stage of sleep. Essential growth hormones are released during this stage.  

During all three stages of NREM sleep, the muscles of the body exhibit tone and movement; individuals often turn in their sleep and reposition themselves in the bed. Although control of core body temperature and regulation of respiration is maintained during NREM, the respiratory rate slows and tidal volume decreases, causing an increase of 2 to 4 mm Hg in PaCO₂. Minute ventilation is approximately 13% to 15% lower in NREM sleep than during wakefulness. Systemic blood pressure may decrease by 5% to 10% during NREM stages 1 and 2 and decrease 8% to 14% during NREM stage 3 sleep. With advancing age, total time spent in stages 1 and 2 progressively increases, whereas stage 3 sleep decreases significantly.

CONCEPT QUESTION 15-4

Explain the difference between NREM stages 1, 2, and 3 with regard to the sleep cycle.

Rapid Eye Movement

REM sleep is associated with a loss of core body temperature regulation, whereas cerebral blood flow and cerebral temperature increase owing to increased brain activity. Systemic blood pressure becomes variable and elevated during REM. These normal physiological effects of REM place patients with preexisting pulmonary or cardiac disease at greater risk for exacerbations of their disease.

EEG tracings during REM strongly resemble the level of brain activity seen in the awake state (i.e. alpha-like, low-voltage, random, high-frequency waves) (see Figure 15-5). As sleep progresses, adults and children cycle back to NREM stages and return to the REM stage three to five times. The time spent in REM normally increases with each cycle for a total of approximately 25% of the sleep period. Electromyography (EMG) shows skeletal muscle tone at its lowest level during the REM stage, suggesting a paralyzed state. This partial paralysis results in a further decrease of the minute ventilation in healthy adults and children, producing a few associated episodes of hypoxemia and hypercapnia, which are normal during REM. Loss of skeletal muscle tone during REM affects pharyngeal muscles; upper airway resistance increases as pharyngeal tissues relax and narrow the upper airway lumen. As previously mentioned, relaxation of the tongue and soft tissues of the oropharynx are the primary cause of increased upper airway resistance, possibly leading to upper airway obstruction. Additionally, REM sleep is associated with heart rate variability and cardiac arrhythmias.

In summary, during normal sleep, an individual dozes into stage 1 NREM and progresses to stage 2 NREM, and then to stage 3 NREM as sleep deepens. During NREM, the brain is in a state of rest while the body is still active and can respond to stimuli. A cycling between stage 1, 2, and 3 NREM continues for 60 to 90 minutes before transitioning to REM, which normally lasts 5 to 30 minutes. Once REM sleep is initiated, brain activity heightens, and dreaming almost always occurs. Partial skeletal muscle paralysis occurs resulting in variability of ventilation, blood pressure, and heart rate. For patients with OSA, the normal progression to REM with partial paralysis results in oropharyngeal soft tissue relaxation and upper airway obstruction. With a loss of airflow and ventilation, oxygen saturation obtained by pulse oximeter (SpO₂) declines, whereas PaCO₂ increases. Consequently, medullary chemoreceptors sensitive to CO₂ are activated and disrupt the onset of REM, “pulling the patient out of REM” and back into stage 1 or 2 NREM sleep; this event is a type of sleep arousal. This sequence is repetitive and fragments sleep; as the patient regains muscular control in the NREM stage, ventilation is restored, and PaO₂, PaCO₂, and pH values are normalized. Recovery of normal arterial blood gas parameters occurs when the subject reestablishes an open airway with a loud snort or gasp, followed by an increased respiratory rate. The individual then slips back into the next NREM stage only to be disrupted from REM sleep repeatedly throughout the night. Patients with SDB commonly lack years of REM sleep; their histories often reveal that they have not dreamed for many years.

Sleep architecture refers to the pattern in which an individual moves back and forth between sleep stages throughout the night. Although the approximate percentage of sleep time spent in the three stages of NREM and the REM stage previously described represents normal sleep architecture, each person has a distinct pattern unique to his or her own sleep cycle. The graphic representation of a patient’s sleep architecture is known as a sleep histogram and is a standard part of every sleep study; it depicts the summative time spent in each phase of sleep at a glance (Figure 15-6). When a patient’s NREM and REM cycles become fragmented on a regular basis, the patient is said to have poor sleep hygiene. Poor sleep hygiene is often due to the lack of a sleep routine or the lack of appropriate sleep cues that signal the body to prepare for sleep, which is typical of the sleep habits of a night shift worker.

CONCEPT QUESTION 15-5

Describe the physiological changes that occur in the body during REM stage sleep.

SLEEP DISORDERS

Approximately 1 in 6, or greater than 50 million, Americans have some form of sleep disorder. More than 84 sleep disorders have been identified and coded by the World Health Organization’s International Classification of Diseases and Related Health
Problems, 10th Revision (ICD-10), which assists health care providers in categorizing sleep disturbances. The ICD-10 coding system classifies diseases, signs and symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or diseases. In the latest edition of ICD-10, sleep disorders are classified under “Mental and Behavior Disorders” in a subcategory called “Behavior Syndromes Associated with Physiological Disturbances and Physical Factors.” The physiology and comorbidities associated with sleep disturbances are complex; a growing number of patients with SDB complain of numerous chronic ailments, including hypertension, depression, heart failure, obesity, learning disorders, and posttraumatic stress syndrome.

There are six broad classifications of sleep disorders with multiple subcategories (Box 15-1). The six major categories include dyssomnias, parasomnias, medical or psychiatric conditions producing sleep disorders, sleep sickness, snoring, and sudden infant death syndrome (SIDS). The largest and most common category of sleep disorders is the dyssomnia group. Dyssomnias (“difficult sleep”) are primarily characterized either as excessive daytime sleepiness (hypersomnolence) or as the inability to fall asleep (insomnia). SDBs in the dyssomnia category include obstructive sleep apnea (OSA), central sleep apnea (CSA), and mixed sleep apnea (MSA).

Obstructive Sleep Apnea

In the United States, the most common SDB in adults is OSA, which affects an estimated 18 million Americans. The etiology of OSA is primarily anatomical, combining the complications of disordered REM physiology with a decreased oropharyngeal lumen. Contributing factors include macrognathia (large tongue), micrognathia (small mandible), deviated nasal septum, and retrognathia (recessed lower jaw). Depending on oropharyngeal anatomy, loss of muscle tone and muscle paralysis during REM sleep may partially or completely obstruct the airway lumen. Complete airway obstruction produces apnea, defined as the absence of ventilation lasting 10 seconds or more. Partial obstruction of the airway causes hypopnea, defined as a 50% reduction in the tidal volume. Apneic and hypopneic events are critical diagnostic markers documented in all sleep studies on the PSG.

During an apnea event, the patient with OSA continues to make ventilatory efforts despite complete airway obstruction. During inspiration, the diaphragms move downward, which generates greater negative intrapleural pressures as the patient tries to inhale. The chest and abdomen move paradoxically, or oppositely, producing a “seesaw” movement. When paradoxical breathing is matched with PSG tracings that document the absence of airflow for at least 10 seconds, an apneic event has occurred. The effect is similar to placing a hand over the nose and mouth of an individual and asking him or her to breathe in as deeply as possible. Full chest and abdominal efforts are apparent, but they produce no airflow. Patients with OSA commonly describe their regular sleeping experiences as episodes
of suffocation (Figure 15-7). The increase in PaCO₂ prompts arousal of the medullary chemoreceptors, which is documented by the EEG; the patient often gasps, is aroused from sleep, and sits up in bed to open the airway. Often the patient does not completely awaken; however, deep REM sleep is disrupted, bringing the patient into stage 1 or 2 NREM sleep. Most patients are unaware that they sit up in bed, gasp, or cough; they simply lay down again, only to repeat the process throughout the night. As soon as the individual returns to REM sleep, the next obstruction begins. Because of anatomy and gravity, the most vulnerable sleeping position for the patient with OSA is supine. Turning to either side or the prone position may significantly decrease the degree of airway obstruction at REM onset.

If airway obstruction is partial, a hypopnea may occur, and the process just described is repeated except that ventilations are shallow rather than absent. Hypopneas frequently cause EEG arousals, and the patient may reposition in the bed before resuming sleep (Figure 15-8). Such arousals, as depicted on the PSG, are known as respiratory effort–related arousals (RERAs) (Figure 15-9). During RERAs, the thorax and abdomen move in a diminished, synchronous manner, although airflow does not appear to be reduced, and the SpO₂ remains stable. However, RERAs usually result in an EEG arousal, and sleep architecture may be disrupted.

OSA occurs most frequently in elderly obese men with a short neck greater than 17 inches in circumference. Heavy snoring that begins soon after falling asleep is the classic picture of a patient with OSA. Typically, the snoring becomes louder and is interrupted by a period of apnea followed by a loud snort or gasp. After reestablishing the airway, snoring returns, and the cycle repeats. The snoring and gasping events may drive the sleep partner from the bed to another room. Risk factors associated with worsening of OSA include a body mass index...
25 or greater, alcohol consumption before bedtime, smoking, nasal congestion at night, and large tonsils. The consequences of uncorrected OSA include decreased physical performance, decreased mental performance owing to a reduced ability to concentrate, psychosocial problems, impotence, decreased libido, hypertension, exacerbation of coronary artery disease, myocardial infarction, heart failure, and stroke. 9,10

Central Sleep Apnea

The second frequently encountered classification of SDB is central sleep apnea (CSA). CSA is characterized by lack of airflow accompanied by the total absence of thoracoabdominal effort for at least 10 seconds. The cause of apnea is not obstruction of the airway but a lack of normal brain activity associated with the physiological drive to breathe. CSA is responsible for only about 10% of all adult SDB but is more common in children and infants. CSA is observed most often during stage 1 NREM or REM sleep. The cause of CSA is varied and complex. It is thought that CSA may be linked to a loss of the neurological control of breathing secondary to injury, stroke, brainstem lesion, encephalitis, neurodegeneration, radiation treatments to the cervical spine region, or congestive heart failure. 10,11,13

Individuals with CSA are most often not obese and have a normal neck circumference. Snoring is not usually present; however, excessive daytime somnolence, or sleepiness, may occur because of frequent awakenings and lack of restorative sleep. Because CSA is closely associated with neurological issues, patients may report symptoms such as difficulty swallowing, a change in the ability to use their voices, or other body weakness or numbness. Any of these neurological symptoms should be immediately brought to the attention of a physician for complete assessment and diagnostic investigation. The most common sign of CSA is the presence of Cheyne-Stokes respirations (see Chapter 11). 9 Figure 15-10 shows a PSG study of a patient with CSA.

Children with Central Sleep Apnea

Although CSA is much less common in children than adults, it is often found in premature infants born before 37 weeks of gestation. CSA is associated with congenital cardiac disorders that cause an elevation of the PaCO₂. As with adults, hypercapnic drive centers in the brainstem may be insensitive or too immature to trigger corrective breathing stimuli to respiratory muscles. Multiple disorders may precipitate CSA in children and infants, including neuromuscular, neurological, metabolic, gastrointestinal, and hematological abnormalities and infections with an accompanying fever. 8,11

Sudden infant death syndrome (SIDS) is thought by some experts to be associated with CSA, but there is no clinical evidence that the two are linked. The Centers for Disease Control and Prevention defines SIDS as "the sudden death of an infant less than one year of age that cannot be explained after a thorough investigation is conducted, including a complete autopsy, examination of the death scene and review of the clinical history." 14 Although the etiology is unknown, most reported SIDS cases occur in the first 6 months of life with a peak incidence
between 2 and 4 months of age. Previously thought to be linked to CSA pathophysiology, SIDS is now believed to have a familial relationship to OSA and positional sleeping and to be associated with families that have a history of apparent life-threatening events. Research during the last decade has linked SIDS to risk factors such as the prone sleeping position, bed sharing, maternal substance abuse, and maternal cigarette smoking. Despite a 38% decrease in SIDS incidence in the United States, it remains the leading cause of death in children younger than 1 year.

**Mixed Sleep Apnea**

As the name implies, mixed sleep apnea (MSA) is a combination of OSA and CSA in the same patient. A PSG study of the patient with MSA typically reveals an initial CSA event followed by an OSA phase (Figure 15-11). During the CSA portion of the MSA sequence, there is no thoracic wall or abdominal effort associated with loss of airflow and declining SpO₂. Following the CSA event, thoracic and abdominal efforts return, but the effort is met with an upper airway obstruction for the remainder of the MSA sequence. As is often the case, support of the collapsing airway through the use of CPAP may eliminate the OSA aspect of the sequence only to allow the CSA component and continued apnea to persist. Many times, as positive airway pressure (PAP) is titrated in the patient with OSA, an underlying CSA episode is discovered when the OSA issue is resolved. Without the PSG diagnostic/titration study, MSA is virtually impossible to confirm. When the OSA component is treated with PAP, the CSA component can be addressed by providing a backup respiratory rate controller on the PAP device. The use of rate controller PAP devices quickly resolves any CSA issues, and the patient can sleep without apneic episodes.

**CONCEPT QUESTION 15-6**

To what major category of sleep disorders do OSA, CSA, and MSA belong?

**CONCEPT QUESTION 15-7**

Explain the difference between an apnea and a hypopnea event.

**CARDIOVASCULAR EFFECTS OF UNTREATED SLEEP-DISORDERED BREATHING**

SDB is a significant risk factor in the development of hypertension and multiple cardiovascular conditions. Effective therapy can eliminate or reverse the detrimental cardiovascular effects of SDB; nevertheless, cardiovascular disorders are increased in these patients either because SDB is undiagnosed or because patients fail to comply with prescribed PAP therapy. The simple act of “breathing while asleep” is key to the restoration of cardiovascular health and a decreased rate of morbidity and mortality.

**Central Hemodynamic Factors**

Regardless of the cause of SDB, apneas and hypopneas cause arterial oxygen desaturation, hypercapnia, fragmented sleep, and sleep arousals. Hypoxia and hypercapnia have a powerful effect on the cardiovascular system. As discussed in Chapter 6, hypoxia and hypercapnia cause pulmonary vasoconstriction. Pulmonary venous constriction impedes blood flow to the left atrium and ventricle and coupled with pulmonary arteriolar constriction increases right atrial and ventricular pressures.

In SDB, variation in respiratory rate interspersed with apneic periods causes PaCO₂ and PaO₂ to vacillate between normal and abnormal values, which produce a physiological “seesaw” dynamic in ventricular pressures. As a result, systemic blood pressure decreases and rebounds with a parallel decrease and increase in heart rate. Cardiac pressures can increase and then decrease to the point that blood flow momentarily stops; the resulting blood stagnation and stasis clotting may lead to potential stroke or myocardial infarction.

During periods of airway obstruction in OSA and MSA, thoracic inspiratory expansion creates large negative intrathoracic pressures, which decrease left ventricular stroke volume and cause a momentary decrease in systemic blood pressure. Blood flow through the already constricted pulmonary vasculature is decreased further, exacerbating cardiac function, as manifested by diminished cardiac output, stroke volume, and systemic blood pressure. When airway obstruction is resolved through sleep arousal or a change in position, stroke volume, heart rate, and blood pressure transiently increase. This physiological “seesaw” of hemodynamics in patients with SDB occurs during each cycle of apnea. Because episodes of apnea commonly result in some level of arousal, sleep is further fragmented as the patient swings between REM and NREM or is awakened by the event.

**Causal Role in Cardiovascular Disease**

Studies have clearly documented the relationship between long-standing SDB and the predisposition for cardiovascular
disease; sympathetic nervous system overactivity is thought to be a critical mechanism in the pathogenesis of hypertension. Repetitive airway occlusion, hypercapnia, and hypoxia, accompanied by vasodilations in intrathoracic pressures elicit autonomic, neural, and humoral responses. A night of fragmented sleep may adversely affect cardiovascular function during waking hours the next day, although daytime breathing is normal. Nocturnal hypoxemia results in endothelial cell dysfunction, producing vasoconstriction and elevated blood pressure during both sleeping and waking hours. Urinary catecholamine levels, an indicator of sympathetic stress and overstimulation, are elevated in patients with untreated SDB, manifesting the persistence of OSA-induced physiological stress throughout the day. Studies have also shown that when patients with OSA are treated with PAP, and sleep apnea is resolved, urinary catecholamine levels return to normal. Hypertension is frequently resolved or diminished through PAP treatment of OSA.

Patients with untreated OSA have a higher risk of hypercoagulability (i.e., increased tendency to develop intravascular blood clots) and subsequent thrombosis than their PAP-treated counterparts. Hypercoagulability mechanisms are still under investigation, but current research suggests changes in clotting factors might be due to the increased number of neutrophils seen in patients with SDB, along with increased catecholamine levels and compromised blood flow. Hypercoagulability increases the risk of coronary artery disease when it coexists with other conditions present in patients with OSA, including obesity, metabolic syndrome, insulin resistance, diabetes, and hyperlipidemia. Fluctuations in blood flow caused by repeated blood pressure surges in OSA result in additional myocardial work, which is associated with left ventricular hypertrophy, often a precursor of adverse cardiovascular events.

**Heart failure**, or systolic dysfunction (ejection fraction <40%), is asymptomatic in about 2% of the general population. Additionally, diastolic dysfunction (failure of the ventricle to fill properly) is a major risk factor in the development of congestive heart failure, or left ventricular failure. Patients with OSA and CSA commonly have coexisting heart failure and its associated increase in mortality rate. Decreased blood flow leads to myocardial ischemia during periods of negative intrathoracic pressure, which may lead to an increased left ventricular afterload, a decreased left ventricular preload, and a subsequent reduction in stroke volume. The use of PAP produces immediate cardiovascular improvement by relieving hypoxia and hypercapnia; the associated improvement in myocardial oxygenation decreases left ventricular end-systolic volume and improves ejection fraction.

SDB and cardiac arrhythmias are also significantly linked, although the association is not well understood. Atrial and ventricular premature ectopic beats are the most common arrhythmias in SDB. Separating cardiac arrhythmias that result from preexisting heart disease from the effects of SDB is difficult; however, successful treatment of SDB seems to decrease arrhythmias. The role of SDB as an independent risk factor for cardiac arrhythmias is yet to be substantiated in the scientific literature.

### CONCEPT QUESTION 15-8

Describe the physiological effects of hypoxia and hypercapnia on the cardiovascular system. What are the cardiovascular consequences of untreated SDB?

### PHYSIOLOGICAL DIAGNOSIS OF SLEEP-DISORDERED BREATHING

#### Polysomnogram

The treatment of SDB begins with a definitive diagnosis based on a PSG sleep study, which is the “gold standard” diagnostic tool in determining the presence of a sleep disorder. The guidelines for conducting and evaluating sleep studies are established by the Association of Sleep Disorders Center and further defined by Medicare Practice Guidelines. The configuration of the various physiological tracings on the PSG display is called a montage (Figure 15-12). The montage for a typical sleep study includes a minimum of 17 channels of physiological tracings. Electrodes are strategically placed on the patient’s head, face, chest, abdomen, and legs to collect the physiological data (Figure 15-13). All data are recorded continuously from “lights off” to “lights on” including four EEG channels to identify NREM or REM sleep stages, two channels recording right and left eye movement, one channel monitoring chin movement, two channels recording right and left leg movement, and nine more channels documenting snoring, nasal and oral airflow, thoracic and abdominal movement, SpO2, heart rate, electrocardiogram (ECG), and body position.

Sleep clinicians must adhere to strict criteria to conduct the PSG study properly and titrate the appropriate pressure needed to resolve apneas and hypopneas. Apneas and hypopneas must be counted during each hour of the sleep study and serve as the basis for certain calculations. The primary index defining the presence of apneas or hypopneas or both is the apnea-hypopnea index (AHI), or the number of hypopneas and apneas per hour of sleep time. For adults, an AHI greater than 5 per hour with a 4% decrease in SpO2 from baseline is considered abnormal and requires treatment. For infants and children, an AHI greater than 1 is considered abnormal when end-tidal PCO2 is greater than 53 mm Hg or SpO2 is less than 92%. Whether SDB is anatomical or neurological, the AHI is the primary criterion on which the diagnosis of SDB is based. Ideally, the prescriptive pressure derived from a PSG study is the PAP in cm H2O pressure required to keep the patient’s airway patent. The goal in titrating PAP is to reduce AHI to 5 or less per hour while the patient is in the supine position. When PAP is correctly adjusted, the patient’s apnea and hypopnea are immediately resolved. If the correct prescriptive PAP is applied, the patient is able to sleep through the night without sleep fragmentation.

The classification of sleep disorder severity involves many factors, but sleep specialists generally agree that the AHI is a measure of sleep apnea severity. An AHI less than 5 is within normal limits. An AHI of 5 to 20 is termed “mild” sleep apnea. An AHI of 20 to 40 is “moderate” sleep apnea. An AHI greater...
The impact of untreated SDB is pervasive and significantly affects quality of life and longevity. SDB also exacerbates preexisting disorders of the cardiovascular, pulmonary, and endocrine systems, accelerating the development of serious health issues.

**Chronic Obstructive Pulmonary Disease**
Patients with chronic obstructive pulmonary disease (COPD) experience progressive dyspnea with advancing lung disease accompanied by right heart failure, or cor pulmonale, secondary to pulmonary vascular hypertension. When these conditions exist in isolation, the impact on the quality of life is significant and debilitating. SDB superimposed on advanced COPD causes patients to develop persistent hypsomnolence, tiredness, loss of ability to concentrate, decreasing SaO₂ levels, and increasing PaCO₂ levels. Their inability to reach REM without upper airway obstruction (SDB), coupled with small airway obstruction leads to the development of respiratory failure and progressive right ventricular failure. The lack of restorative sleep accelerates muscle fatigue, which is critically detrimental to the ventilation of the hyperinflated COPD thorax.

**Cardiovascular Disorders**
Patients with concurrent SDB and cardiovascular conditions are at high risk for a rapid and progressive decline in cardiovascular function. OSA alone causes swings in central hemodynamic pressures; this occurs as left ventricular stroke volume decreases during inspiratory efforts against an obstructed upper airway followed by a surge in systemic arterial pressure when breathing is restored. Peripheral, cerebral, and pulmonary circulations are thus subjected to periods of hypertension and hypotension. SDB is highly correlated with congestive heart failure in the population at large. The large ventricular afterload produced by OSA events has been shown to depress cardiac function in patients with preexisting congestive heart failure. Additionally, untreated severe sleep apnea has been shown to be associated with stroke, coronary artery disease, cardiac arrhythmias, and pulmonary hypertension—which leads to right ventricular failure.

**Insulin Resistance**
Since 1993, studies have shown the relationship between glucose dysregulation and OSA. A growing body of evidence suggests OSA can independently impair insulin sensitivity resulting in insulin resistance. A newly described metabolic condition reaching near-epidemic proportions in Western society, “syndrome Z,” has many features common to OSA, including obesity, hypertension, hyperlipidemia, and insulin resistance. Since PAP is the most common treatment for OSA, the question of the effect of PAP on insulin sensitivity is an unanswered question. In a group of 40 patients with OSA, Harsch et al. showed that insulin sensitivity improved 18% after 2 days of PAP treatment and improved 31% after 3 months of treatment. It has been theorized that acute periods of hypoxic stress and sleep fragmentation may impair glucose homeostasis, leading to insulin resistance and type 2 diabetes.

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**CLINICAL FOCUS 15-3**

**Sleep-Disordered Breathing and Comorbidities**

The impact of untreated SDB is pervasive and significantly affects quality of life and longevity. SDB also exacerbates preexisting disorders of the cardiovascular, pulmonary, and endocrine systems, accelerating the development of serious health issues.

Describe the diagnostic process of determining the presence of SDB.
Figure 15-12 Polysomnogram montage of physiological data. (From Wilkins RL, et al: Clinical assessment in respiratory care, ed 6, St Louis, 2010, Mosby.)

Figure 15-13 Patient with sleep-disordered breathing ready for a sleep study. (From Wilkins RL, et al: Clinical assessment in respiratory care, ed 6, St Louis, 2010, Mosby.)
**CLINICAL FOCUS 15-4**

**Pharmacology of Sleep-Disordered Breathing**

Effective drug therapy for SDB is yet to be discovered. SDB refers to sleep disorders in which normal breathing is affected during sleep, and the specific pharmacology to relieve sleep apnea is lacking. Upper airway obstruction is the primary etiology of sleep apnea (OSA and MSA); a specific pharmacological agent that targets receptor sites, shrinks soft tissues, and reestablishes airway patency has not been found. Both serotoninergic and serotonin receptor antagonist drugs have been tested for effectiveness in reducing SDB, but none of these drugs have been shown conclusively to reduce sleep apnea. At best, sleep pharmacology may be helpful for some individuals as an adjunctive therapy when combined with PAP treatment.  

Ventilatory stimulants, including medroxyprogesterone, thyroxine, acetazolamide, and theophylline, have not been shown to benefit patients with moderate to severe OSA. In some cases, the induced ventilator efforts appear to increase chest wall work and airway collapse secondary to increased intrathoracic pressures. Opioid antagonists and nicotine have also been used in attempts to stimulate ventilation in patients with OSA with minimal changes in the number of apneic events and an undesirable increase in sleep fragmentation. However, some studies have shown that ventilatory stimulation may be of some use for patients with CSA in the absence of upper airway obstruction.

Studies of psychotropic agents, such as protriptyline, serotonin precursors (L-tryptophan), and benzodiazepines, have shown a varying impact on patients with SDB and a reduction in AH1 and improved sleep in a small percentage of subjects but not to a statistically significant level. Antihypertensive agents, such as hydralazine, metoprolol, and cilazapril, have been used to decrease sympathetic tone to improve SDB. Although antihypertensive agents were found to decrease the number of apnea events, the mechanism is thought to be due to a REM-suppressing effect.  

Sleep pharmacology for non-SDB categories of sleep disorders is of significant importance. As popularly advertised, sleep medications address issues of insomnia rather than SDB. Insomnia often may be an accompanying symptom of excessive daytime sleepiness that results from OSA during the night. The patient becomes sleepy during the day, naps frequently, and has difficulty attaining sleep onset or experiences prolonged sleep onset at night, which is interpreted as insomnia. Agents such as modafinil (Provigil) taken once in the morning may reduce the excessive daytime sleepiness of OSA, narcolepsy, and shift work sleep disorder by maintaining alertness during the day. For other patients with SDB, the use of PAP treatment may lead to increased sleep efficiency, decreased excessive daytime sleepiness, and abatement of insomnia without adjunctive medication. Sleep pharmacology for primary sleep disorders is presented in Box 15-2.
Split-Night Polysomnogram Study

Patient Assessment
A 55-year-old man with complaints of snoring, apnea, daytime fatigue, restless legs, shortness of breath, nocturnal awakenings, and severe hypersomnia was referred to a sleep center for a diagnostic PSG and PAP titration, if indicated. Patient medical history includes hypertension, allergies, asthma, gastroesophageal reflux disease (GERD), tonsillectomy, and adenoidectomy. The patient’s height of 69 inches and weight of 240 lb produces a calculated body mass index of 35.4. The patient is a former 20-pack-year smoker who quit approximately 10 years ago; he consumes 5 to 10 caffeinated beverages daily to maintain alertness, and consumes 3 to 4 alcoholic beverages each evening. He has no regular exercise routine. Neck circumference is 18.5 inches with a Mallampati classification of class III.

A complete PSG digital sleep system diagnostic was performed using the international 10- to 20-electrode placement for recording EEG, electrooculography near the eyes, EMG from the chin, ECG, respiratory effort, oximetry, body position, airflow, snoring monitor, pulse rate, and limb movement channels. Following full PSG hook-up, the patient was provided specific education regarding PAP titration should the diagnostic test meet standard guidelines for intervention.

Pretreatment Polysomnogram Summary
The patient’s total sleep efficiency was 82.9% with 0.0% REM sleep, 22.9% stage 1 sleep, 76.6% stage 2 sleep, and 0.6% stage 3 sleep. There were 45 obstructive apneas, 0 central apneas, and 0 mixed apneas with 124 hypopneas with SpO₂ desaturations 3% or greater and 6 RERAs. The apnea index was 30.9, and the AHI was 115.9 with a respiratory disturbance index (RDI) of 118.6. The lowest SpO₂ recorded was 80% with 15.9 minutes of desaturations from 81% to 90%. The mean SpO₂ during sleep was 93%. Following full PSG hook-up, the patient was provided specific education regarding PAP titration should the diagnostic test meet standard guidelines for intervention.

Discussion
Multiple symptoms relevant to OSA are present in the patient history, including snoring, apnea, daytime fatigue, restless legs, shortness of breath, nocturnal awakenings, and hypersomnia. Additionally, the presence of obesity, use of caffeinated beverages during the day to maintain alertness, use of alcohol before bedtime, a neck circumference greater than 17 inches, and a Mallampati classification of class III complete a scenario common to patients with OSA. The pretreatment PSG study shows no REM sleep with 22.9% stage 1 sleep, 76.6% stage 2 sleep, and 0.6% stage 3 sleep. This sleep architecture suggests very poor sleep quality with no REM and most of the sleep time spent in stage 2 sleep with negligible time spent in restorative stage 3 sleep. SpO₂ values indicate mild hypoxia during sleep with 220 leg movements during sleep that likely cause sleep arousals. The apnea index was 30.9 apneic events per hour; a normal apnea index is less than 5 events per hour. An apnea index of 30.9 equates to having an apnea episode of greater than 10 seconds every 30 seconds of sleep. Sleep fragmentation is quite severe.

After qualifying for PAP titration, the patient was fitted with a CPAP mask at a beginning pressure of 5 cm H₂O. After returning to sleep, the sleep technician began to titrate the CPAP incrementally while observing the number of apneic events. When a CPAP of 12 cm H₂O was reached, the sleep technician noted there were no apnea events and no hypopnea events. In the supine position, the likelihood of airway obstruction is greatest, but a CPAP of 12 cm H₂O held the upper airways open and prevented obstruction by the tongue or soft tissues. As a result, the patient experienced “REM rebound” spending 40.2% of the sleep time in REM sleep, 3.8% in stage 1 sleep, 42.8% in stage 2 sleep, and 13.1% in stage 3 sleep. Sleep efficiency was increased to 92%, and the prescription pressure of 12 cm H₂O was verified through an AHI of 0.0.
ANATOMICAL TREATMENT OF SLEEP-DISORDERED BREATHING

In theory, the treatment for OSA is simply the maintenance of a patent airway during sleep. During REM, the relaxation of oropharyngeal muscles and the tongue partially or completely obstruct the airway. Options for treating SDB include PAP therapy, oral appliances, positional solutions, and airway enlargement procedures.

Positive Airway Pressure

SDB caused by soft tissue obstruction of the oropharynx can be immediately resolved without surgery or pharmaceuticals through the use of properly administered PAP therapy. Judicious titration of PAP is required to identify the precise pressure that alleviates sleep apnea, snoring, and hypopneas. Each titration sleep study is individually tailored to meet the specific needs of the patient, which are related to the degree of obstruction present. PAP treatment begins with a properly sized and fitted mask. With more than 50 brands and types of masks available, the sleep clinician must be an expert in finding the properly designed mask that accommodates each patient’s particular facial features and anatomical structures. The clinician chooses a nasal or oronasal mask after assessing the patient’s facial anatomy. If the patient requires high PAPs, nasal masks are not suitable, and an oronasal or full facial mask is needed to provide the appropriate pressure without leaking. Additionally, patient comfort is a critical factor affecting continued patient use or compliance with PAP therapy. Over-tightening the mask can cause leaks or patient discomfort and is usually a sign of improper sizing. Without the proper mask fit, applied pressures do not reliably keep the airways patent (Figure 15-14). 21

More than 50 models of PAP, CPAP, and BIPAP generators exist, and it is beyond the scope of this chapter to detail their differences. Various PAP modes and delivery systems are available to meet specific patient needs, including CPAP, BIPAP, average volume assured pressure support (AVAPS), auto-CPAP, BIPAP-autoSV (support ventilation), BIPAP S/T (spontaneous/timed), volume positive airway pressure (VPAP), Smart CPAP, and demand positive airway pressure (DPAP). Each mode has specific advantages that must be matched to the patient’s specific SDB needs. For this reason, a titration sleep study must be completed in the sleep laboratory under direct supervision of a trained clinician to allow titration and adjustment of PAP equipment during the night. Some physicians and medical equipment companies attempt to provide treatment for documented SDB without a titration study, but patient compliance and continued use is threatened by this practice. Current manufacturers of PAP devices provide multiple-mode devices that are small, quiet, and efficient. Patients using PAP must be able to transport the device and all associated equipment safely when traveling away from home; the size of the device has a significant impact on patient compliance during travel. 5,11,21

As previously mentioned, the length of the REM stage normally increases through the night with the longest phase of REM typically just before waking. Because the REM state represents skeletal muscle paralysis and decreased ventilation efforts, the PAP device must be adjusted to meet all ventilation needs and supply complete support of flaccid or collapsing soft tissue in the oropharyngeal cavity. With the CPAP or BIPAP adjunct in place with the appropriate PAP device, the PSG titration study documents the PAP setting required to stent open the airways while the patient transitions through each stage of sleep. PAPs are adjusted in each sleeping position until all sleep parameters are normalized and documented with an AHI less than 5. Because the supine sleeping position is the patient’s most vulnerable position with regard to airway collapse, the therapist must ensure the PAP is titrated to the correct pressure during REM while the patient is supine. When this pressure is determined, all other stages of sleep and sleeping positions are likely to be supported with enough PAP for uninterrupted sleep. 10

Following the titration sleep study in which PAP successfully normalizes the AHI, the prescription pressure and supporting documentation are reported back to the ordering physician, and the patient is referred to a durable medical equipment (DME) company to purchase a home PAP device. The respiratory therapist from the DME company properly fits the patient with a PAP adjunct, instructs the patient how to use the PAP equipment, and provides patient education relevant to the type of SDB present. PAPs are preset by the DME therapist according to the physician’s order; the therapist instructs the patient in monitoring the mask to ensure proper and active function of the device. The device should be set to pressurize to the prescriptive pressure automatically. Both the physician and the DME company are responsible to provide patient follow-up on PAP compliance and resolve any issues affecting treatment adherence.

Oral Appliances

Known also as dental appliances, oral appliances are acrylic devices that fit over the upper and lower teeth similar to a mouth guard. An oral appliance is designed to move the lower jaw or mandible, slightly forward. Because the tongue is attached to the mandible, the tongue is also advanced forward to provide a larger oropharyngeal lumen. A dentist experienced in the treatment of snoring and sleep apnea must customize the oral appliance for each patient. Two of the oral appliances in current use are the mandibular advancement device (MAD) and the tongue retaining device (TRD). The MAD device is designed with an adjustable mechanism that allows the patient to advance the mandible gradually daily, eventually increasing the oropharyngeal airway opening. The TRD fits around the tongue and holds it forward by means of suction; it is used most often by patients with lower dentures that are removed at night or by patients in whom the mandible cannot be effectively advanced. Both devices require the patient to be able to breathe through the nose adequately; otherwise, the oral appliance will not be tolerated (see Figure 15-15A and B). Although oral appliances are an option for the treatment of OSA, results are marginally successful, and these appliances are effective only in patients diagnosed with mild OSA. 22,23
Figure 15-14  A-C, Continuous positive airway pressure and bilevel positive airway pressure masks.

Figure 15-15  Mandibular advancement device (A) and tongue retaining device (B) oral appliances.
Positional Therapy

Given that gravity worsens sleep apnea in the supine position, one approach to reducing apneic episodes is the avoidance of the supine position. Individuals normally move and reposition themselves through the night during each cycle of NREM sleep. The goal of positional therapy is to maintain a nonsupine position. The “snore ball” is a hard plastic sphere the size of a tennis ball that is sewn into the middle of the back of pajamas or a tee shirt worn by the patient at night. The supine sleeping position is discouraged by the uncomfortable ball positioned over the spine. In theory, the discomfort prompts the sleeping patient to roll to either side and out of the supine position.

Another approach to positional therapy involves pillows registered by the U.S. Food and Drug Administration (FDA) and specifically designed to prevent snoring and mild sleep apnea. These pillows are designed to reposition the neck so that the oral airway is more likely to remain patent. The foam wedge-shaped pillows are designed at angles of various degrees and presumably reduce positional collapse of the airways and reduce apneas and snoring. However, the manufacturer recommends that the patient sleep in the supine position on the positional pillows. At best, positional therapy may prove beneficial in reducing the number of apneic periods and snoring intensity in mild sleep apnea, but it has not been found to be effective in eliminating moderate or severe obstructive apnea.24

Airway Enlargement

The treatment of last resort for obstructive SDB is surgical enlargement of the oropharyngeal cavity to provide a more patent airway. Surgery entails the reduction of soft tissue structures and may be effective in some OSA conditions, but the patient should be fully aware of potentially adverse outcomes. A common surgical procedure for sleep apnea is uvulopalatopharyngoplasty (UPPP), which involves removal or shortening of the uvula, removal of the tonsils and adenoids, and excision of portions of the soft palate or roof of the mouth; the goal is enlargement of the airway lumen. The benefits of UPPP are controversial. Although 90% of patients experience symptomatic improvement from habitual snoring, studies have shown that only 41% to 66% of patients undergoing UPPP see improvement or elimination of OSA, with results worsening over time.25 The efficacy of UPPP cannot be predicted, and nonsurgical options should be considered before resorting to the procedure. Children with OSA should be assessed for enlarged tonsils and adenoids. Simple tonsillectomy/adenoidectomy has been effective in relieving snoring and sleep apnea in children.26,27

Other surgical treatment of OSA includes midline glossectomy and lingualplasty, in which part of the tongue is removed to enlarge the oral cavity. In addition, maxillomandibular advancement (MMA), which moves the mandible anteriorly, has been successful, but this procedure requires the jaw to be broken, extended, and wired shut until the mandible heals.28 Radiofrequency tissue ablation (RFTA) was approved by the FDA in 1998 and is used to shrink the size of the tongue and palate with high-frequency waves; the procedure can be performed in the physician’s office.29 The most effective surgical treatment of OSA is a tracheotomy, although it is deemed unacceptable by most patients. When a tracheotomy is performed for this purpose, the stoma is closed or plugged during the day and unplugged during the night.

The least invasive method to promote airway enlargement is weight loss. Multiple studies have shown that when patients with OSA lose a significant amount of weight, the lumen of the oropharyngeal airway is increased, and the reduced weight on the chest, airways, and abdominal cavity decreases work of breathing during sleep. Obese patients who lose more than 100 lb may see a reduction in PAPs needed; in some cases, OSA may completely resolve with weight loss.30 OSA is an anatomical issue not always associated with obesity; thin individuals may also experience OSA because of their airway anatomy.

CONCEPT QUESTION 15-10

Discuss the four major categories of SDB treatment, and identify the most common method of treatment.

POINTS TO REMEMBER

• The hypothalamus is responsible for the neurocontrol of sleep by controlling the timing of the sleep cycle through the suprachiasmatic nucleus and the ventrolateral preoptic nucleus.
• Humans become obligate nose breathers during sleep, and the upper airways must remain open to accommodate normal restful sleep.
• The Mallampati score can be used to assess whether the oropharyngeal anatomy is too crowded to allow proper ventilation during sleep.
• During NREM stages 1, 2, and 3, the brain enters a restful state while the skeletal muscles respond to stimuli with movement. The NREM cycle lasts 60 to 90 minutes with transitions between all three stages.
• In the REM stage, brain wave activity increases in intensity to a level that resembles the awake state. Skeletal muscles become partially paralyzed during REM, and the loss of muscle tone allows soft tissues to relax.
• REM usually lasts 5 to 30 minutes and is characterized by rapid eye movement and dreaming. During normal sleep, NREM and REM cycle three to five times per night.
• Relaxation of muscles in the oropharyngeal airways can result in the tongue and other soft tissues obstructing airflow producing a period of apnea or hypopnea. During this event, the PaO2 decreases and the PaCO2 increases until the patient has an EEG arousal, gasps, and moves until the airway can be reestablished.
• SDB is classified as dyssomnias; OSA, CSA, and MSA are the most common sleep disorders in the United States.
• OSA is the most common form of SDB and is characterized by snoring and gasping with numerous and prolonged periods of apnea and hypopnea. OSA imposes a physiological strain on the cardiovascular system with a significant
predisposition for hypertension, cardiac arrhythmias, heart failure, potential myocardial infarction, and stroke.

- CSA manifests with periods of apnea without respiratory effort or obstructed airways. A lack of neurocontrol of breathing is thought to be responsible; CSA is also seen in premature infants and children.
- The PSG is considered the “gold standard” diagnostic tool to determine the presence of a sleep disorder. Physiologically data are collected and scored by the sleep technician or respiratory therapist before being interpreted by a qualified sleep physician.
- The goal of treatment for SDB is to establish open airways during sleep. Maintaining an open airway can be accomplished through PAP devices, oral appliances, positional therapy, and airway enlargement options.

References